Disappearing Saccular Intracranial Aneurysms: Do They Really Disappear?

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Summary

Evolution and natural history of cerebral aneurysms is a dynamic process. Spontaneous regression in size or complete disappearance of an aneurysm is a known phenomenon, more commonly noted in giant intracranial aneurysms. However, reappearance or regrowth of such aneurysms is rare with few anecdotal reports. We report a series of four cases including one giant aneurysm, which either disappeared or regressed on sequential angiograms. Regrowth or reappearance of two of these previously disappeared or regressed aneurysms was noted and endovascularly treated while the other two cases are being followed up. The decision to follow up was crucial considering the nature of the aneurysms to change in morphology under the influence of various hemodynamic factors.

Introduction

Cerebral aneurysm most commonly presents as subarachnoid hemorrhage due to rupture. Spontaneous disappearance of an intracranial aneurysm is known and more commonly seen in giant aneurysms [1]. Many factors for spontaneous disappearance have been discussed in literature; reappearance has been a relatively rare occurrence. We report a series of four cases of cerebral aneurysms which disappeared (three) or regressed (one) and reappeared on sequential angiograms and were treated by endovascular coiling.

Case Reports

Patient 1:

A 32-year-old, non-smoker, non-diabetic, and normotensive man presented with a history of sudden onset headache for seven days, tonic-clonic movements of the left upper limb for two days and diplopia for one day. Clinical examination revealed mild papilledema with minimal weakness of left upper limb. CT revealed well-defined hyperdense lesion in the suprasellar cistern on right side with homogenous intense enhancement (figure 1A,B). CT angiography showed a saccular aneurysm arising from the right posterior communicating artery (figure 1C).

Digital subtraction angiography of the right internal carotid artery (ICA) revealed a saccular aneurysm arising from posterior communicating artery measuring 14.5 x 10.7 x 7.5 mm with neck of the aneurysm measuring 4 mm (figure 1D).

The angiogram also revealed spasm of the right ICA and stasis of the contrast within the aneurysm sac on delayed phase (figure 1E). Baseline angiogram on the scheduled day of intervention seven days after the first angiogram revealed near total disappearance of the aneurysmal sac with rest of the intracranial vessels appearing normal without evidence of spasm (figure 1F). These findings were considered to be suggestive of spontaneous thrombosis of the aneurysm and coiling of the aneurysm was deferred.

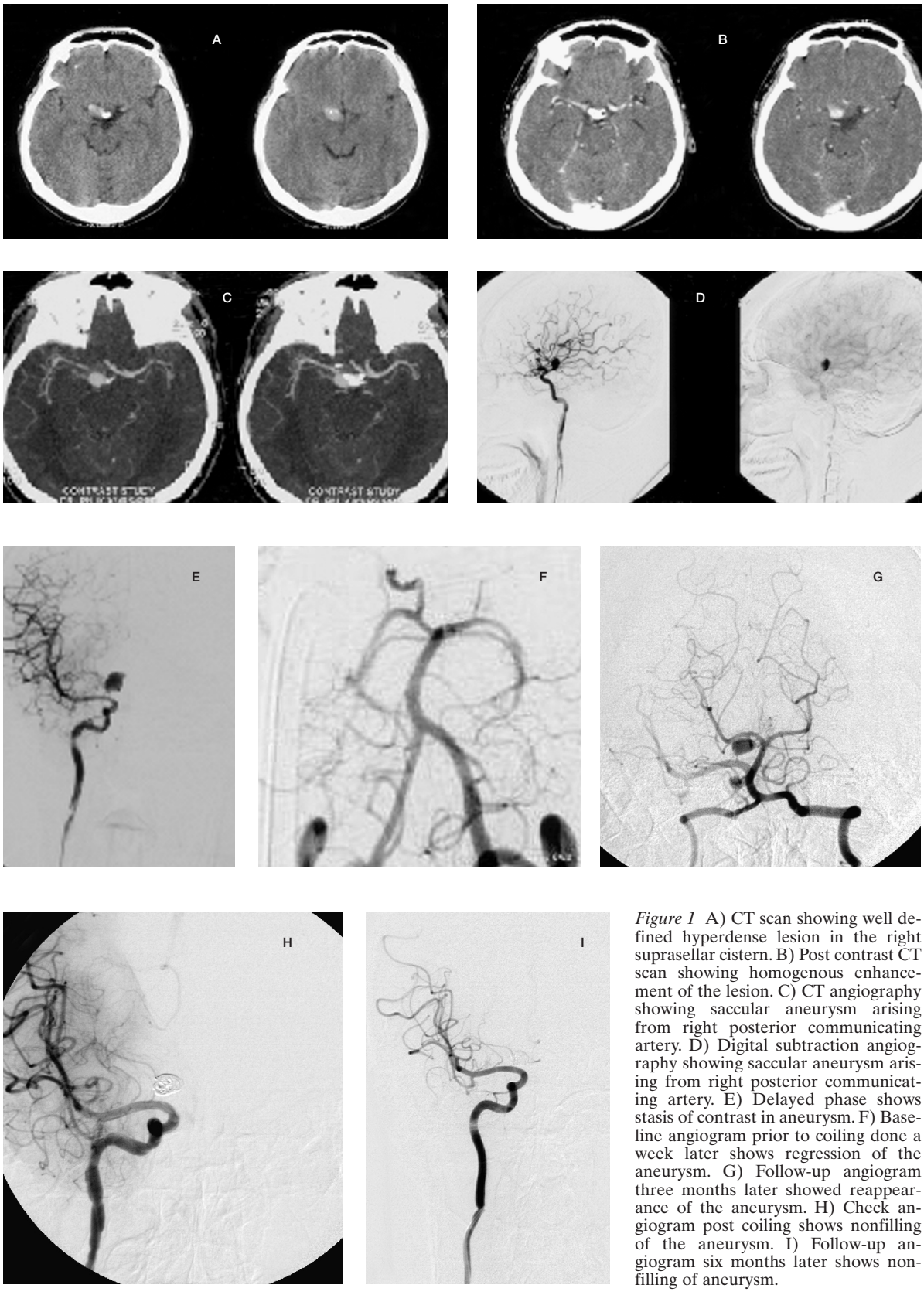

Figure 1 A) CT scan showing well defined hyperdense lesion in the right suprasellar cistern. B) Post contrast CT scan showing homogenous enhancement of the lesion. C) CT angiography showing saccular aneurysm arising from right posterior communicating artery. D) Digital subtraction angiography showing saccular aneurysm arising from right posterior communicating artery. E) Delayed phase shows stasis of contrast in aneurysm. F) Baseline angiogram prior to coiling done a week later shows regression of the aneurysm. G) Follow-up angiogram three months later showed reappearance of the aneurysm. H) Check angiogram post coiling shows nonfilling of the aneurysm. I) Follow-up angiogram six months later shows nonfilling of aneurysm.

Follow-up angiography after three months demonstrated reappearance of the aneurysm with dimensions of the aneurysm almost equal to the initial angiographic study (figure 1G); the volume of the aneurysm measured about 1020.5 mm^3 (volume was obtained by considering aneurysm shape to be ellipsoidal, according to the formula: V= 4/3 pi [a/2][b/2][(a+b)/4]; a & b being the largest vertical and horizontal diameters of the aneurysm and oriented perpendicularly [2].

The aneurysm was coiled in the same sitting with GD coils (Guglielmi Detachable Coils; Boston Scientific Corp, Fremont, USA). Check angiogram showed total occlusion of the aneurysmal sac (figure 1H). Follow-up angiogram done after six months revealed complete occlusion of the aneurysm (Figure 1I).

Patient 2

A 29-year-old man was referred with a basilar artery aneurysm. Two earlier attempts at clipping the aneurysm surgically at another centre had been unsuccessful. A diagnostic angiogram at our centre after the second operation revealed an aneurysm that was about 3.2 x 3.1 x 3 mm in size (volume-15 mm^3) with a neck that measured 2.5 mm arising from the basilar artery just below the origin of the superior cerebellar arteries (figure 2A,B). The patient consented to coiling three weeks later. Baseline angiogram prior to the coiling revealed that the aneurysm had considerably regressed in size and now measured about 2 x 1 mm (figure 2C,D). These findings were considered to be suggestive of spontaneous thrombosis of the aneurysm and coiling of the aneurysm was deferred. Follow-up angiogram after two months revealed that the aneurysm had increased in size more than the original dimensions and now measured 4 x 4.6 x 3 mm with a neck of 2.3 mm (figure 2E,F) and it was decided to coil the aneurysm. A 6F guiding catheter was placed in the left vertebral artery and the aneurysm was treated with GD coils. Check angiogram showed near total occlusion of the aneurysm (figure 2 G-I). The patient is on follow-up.

Patient 3

A 27-year-old man presented with complaints of severe headache, pain in the left eye and progressive loss of vision in the left eye for a month. On examination he had left lateral rectus palsy and hypoaesthesia in the distibution of left V1 territory. CT scan revealed a well defined hyperdense lesion in the left paracavernous region which showed enhancement on post contrast images (figure 3A). MRI revealed a heterointense lesion which was predominantly hyperintense on T2-weighted images with areas of hypointensity. The flow void of the left internal carotid artery appeared to be ending in the lesion and could not be separated from it (figure 3B). MR angiogram revealed that the cavernous segment of the left internal carotid artery was narrowed and ended in a large hyperintense paracavernous lesion (figure 3C) which suggested that the lesion was a giant aneurysm arising from the cavernous segment of left internal carotid artery ICA).The aneurysm measured 37.2 mm x 35.7 mm x 36.5 mm. A decision to occlude the left ICA which was the parent artery was taken as the aneurysm was a large aneurysm and as visualised on MRI scans the parent artery appeared to be in severe spasm and its distal end incorporated in the aneurysm. Baseline angiogram which was done about four weeks later revealed a total block of the left internal carotid artery just distal to its origin (figure 3D).The left intracranial circulation was being supplied across the Circle of Willis by the contralateral side (figure 3E). Thus there was a spontaneous thrombosis of left internal carotid artery including the aneurysm. The patient is on follow-up.

Patient 4

A 55-year-old woman presented with complaints of severe headache which was localised more to the occipital region. On examination the patient had neck rigidity without any focal deficits. CT scan showed hyperdensity in the perimesencephalic and prepontine cisterns suggestive of subarachnoid haemorrhage (figure 4A). Angiogram revealed a small 2 x 2 mm aneurysm arising from the basilar artery just below the origin of the superior cerebellar arteries. The fundus was pointing posteriorly and the neck measured about 2 mm and this was better seen on the rotational 3D angiogram (figure 4B,C).

The volume of the aneurysm was 5 mm^3. It was planned to treat the aneurysm by endovascular coiling. Two weeks later when the patient was taken up for the procedure, check angiogram revealed no evidence of any aneurysm (figure 4D,E). The patient is currently on follow-up.

Discussion

Fodstad and Liliequist found spontaneous disappearance of ruptured aneurysms on repeat angiography in 3% of patients with subarachnoid hemorrhage [3]. Spontaneous thrombosis is a well-known phenomenon in the natural history of intracranial aneurysms and considered a cause for such disappearance. While few cases of ruptured aneurysms (about 1-2%) show evidence of spontaneous and complete thrombosis on subsequent angiography [4], it is more common in giant intracranial aneurysms, occurring in 48-76% of cases [1].

Various biophysical and dynamic parameters related to aneurysm sac and blood flow have been suggested to contribute to spontaneous thrombosis of intracranial aneurysms.

1) The stasis of blood in the aneurysm sac: Increase in intracranial pressure after subarachnoid hemorrhage results in a decrease in cerebral blood flow and localized spasm of the parent artery harboring the ruptured aneurysm are two important hemodynamic alterations resulting in stasis of blood in the parent artery and in the aneurysmal sac.

2) Aneurysm sac volume to orifice ratio: Black and German claimed in their experimental animal model of aneurysms that a ratio of sac volume (in cubic millimeters) to orifice area (in square millimeters) of greater than 28:1 was associated with spontaneous thrombosis [5].

3) Experimental models have shown that flow in large aneurysms is complicated by stagnation of blood, increased blood viscosity and slow flow which may lead to thrombosis [6].

4) Endothelial injury due to turbulent blood flow may lead to increase in platelet deposition, platelet aggregation and thrombus formation [7].

5) Stasis of contrast material in the aneurysmal sac is also an important factor in thrombus formation. The relation between contrast media and clotting mechanism is poorly understood [8]. Contrast media are considered contact activators of coagulation, the presence of which leads to red cell and endothelial damage [9]. The release of tissue based thrombogenic substances is considered to induce thrombosis [10].

Many of the factors that are purportedly responsible for spontaneous thrombosis may be noted in patient 1. The giant aneurysm size, sac volume to neck ratio which was 81.25:1, stagnation of contrast late into the venous phase noted within the aneurysm and stasis of contrast material in the sac might have led to endothelial injury with activation of the clotting process. Persistence of endothelium is necessary for persistence or growth of the aneurysm [11] and endothelial injury or denudation might cause thrombosis. Ionic contrast media which was used in this case (also in cases 3 and 4) are known to cause more endothelial injury than nonionic contrast media [12,13]. Further, though there was no evidence of rupture of the aneurysm on CT, there was evidence of vasospasm on the angiogram.

Patient 3 had an aneurysm of the internal carotid artery which was large, had a large sac volume and stagnation of blood was also evident. There was spasm of the parent artery which could have been due to the mass effect of the giant aneurysm leading to stagnation of flow. This has been reported previously in cases of giant intracavernous internal carotid artery aneurysms which thrombosed along with parent artery [14,15].

Table 1 **Haemodynamic factors in the four cases.**

	Age/ Sex	Factors noted on initial presentation				
		Stasis of blood /contrast	Volume of aneurysm: orifice area	Contrast medium used	Vasospasm	SAH/ Rupture
1.	32/M	present	81.25:1	ionic	present	+
2.	29/M	present	3:1	non ionic	absent	-
3.	27/M	present	*	ionic	present	-
4.	55/F	absent	1.4:1	ionic	absent	+

**- orifice could not be measured*

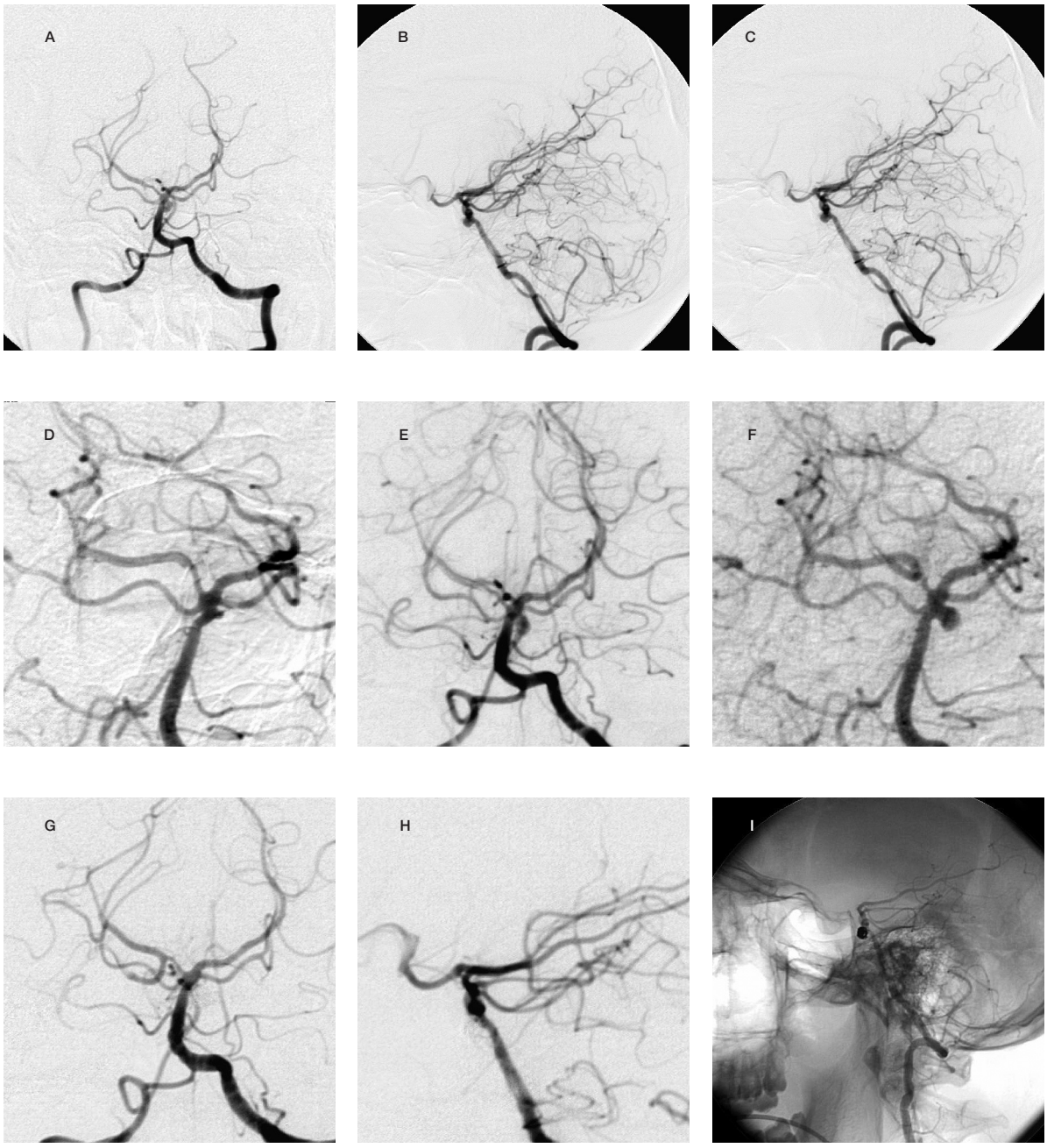

Figure 2 A,B) AP and lateral views showing saccular aneurysm arising from distal basilar artery just below origin of superior cerebellar arteries. C,D) Baseline angiogram prior to coiling three weeks later shows regression in size of aneurysm on AP and Lateral views. E,F) Follow-up angiogram two months later shows reappearance of aneurysm similar in dimensions to the first angiogram. G,H) Post coiling check angiogram shows absence of filling of the aneurysm. I) Coil mass noted in situ.

It has also been pointed out that thrombosis of giant aneurysms is not only due to intraluminal factors but also due to the vessel wall itself. Adventitial inflammation and the vasa vasorum are known to play an important role in the evolution and pathogenesis of these aneurysms [16]. Subsequent or follow-up MR imaging which could not be performed in our subjects would probably have helped in further elucidating the role of the wall of the aneurysm in its pathogenesis. However, the aneurysms in cases 2 and 4 were small in size. Sac volume to orifice area

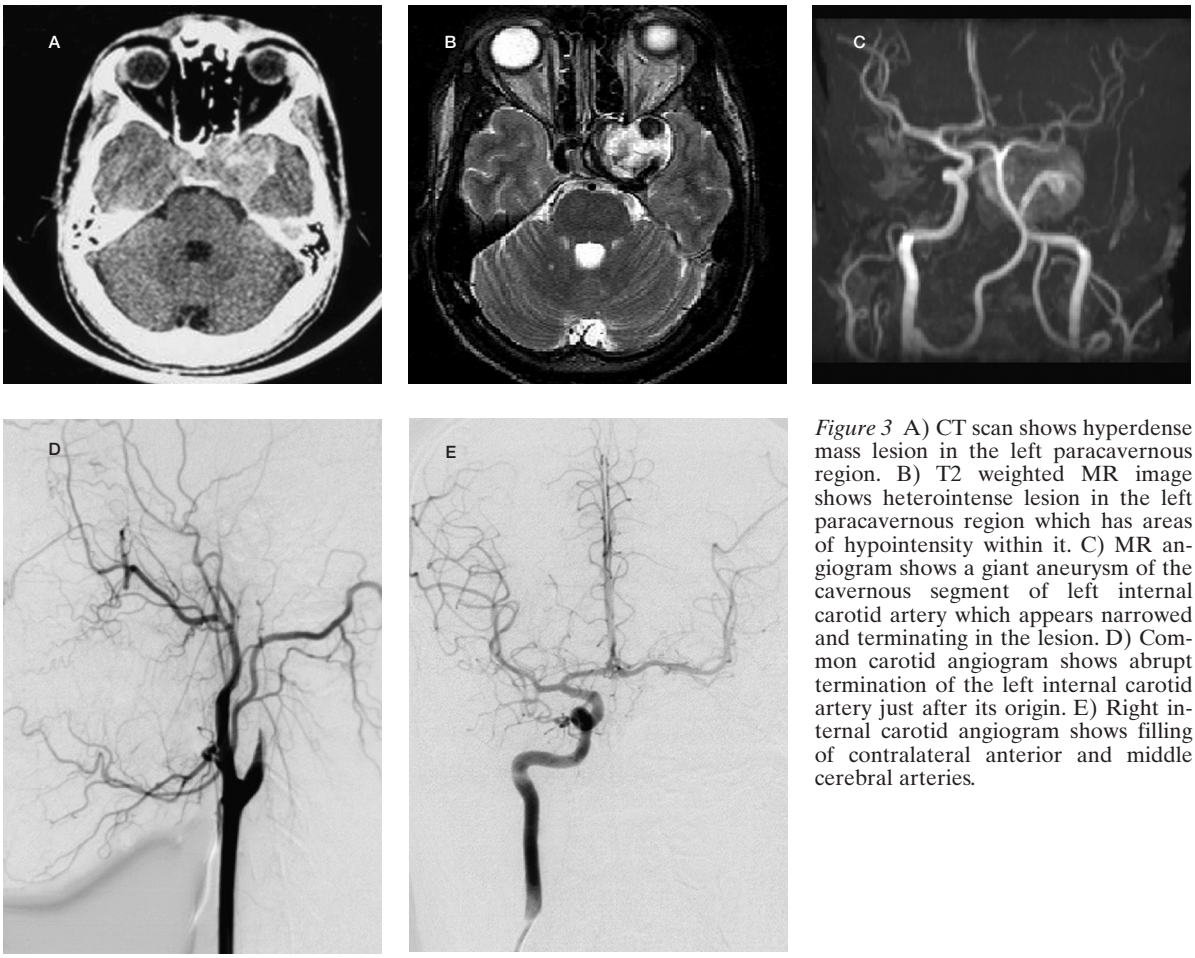

Figure 3 A) CT scan shows hyperdense mass lesion in the left paracavernous region. B) T2 weighted MR image shows heterointense lesion in the left paracavernous region which has areas of hypointensity within it. C) MR angiogram shows a giant aneurysm of the cavernous segment of left internal carotid artery which appears narrowed and terminating in the lesion. D) Common carotid angiogram shows abrupt termination of the left internal carotid artery just after its origin. E) Right internal carotid angiogram shows filling of contralateral anterior and middle cerebral arteries.

was also within normal limits in both cases.The angiograms also did not reveal significant vasospasm. However CT scan showed evidence of SAH in patient 4 which suggested that the aneurysm had probably ruptured.

Chow et Al[17] suggest that in cases with SAH, reasons for thrombosis include vasospasm or treatment with epsilon aminocaproic acid. While, such haemodynamic alterations caused by vasospasm could lead to stasis and thrombosis[17] there was no such obvious changes in patients 2 and 4. While, non-ionic contrast medium used in case 2 could be an incriminating factor, the cause for disappearance of the aneurysm in case 4 remains unexplained. Non ionic contrast media have been shown to have a procoagulant effect and have a higher propensity for thrombosis[8,9,18].

Unlike ionic contrast media which cause endothelial injury, platelet aggregation and fibrin polymerisation appear to be more with nonionic contrast media[18,19]. Recanalisation of thrombosed aneurysms which have been treated by endovascular methods is a known phenomenon. Low thrombogenic nature of the embolic material, incomplete occlusion of the aneurysm due to loose filling and residual neck have been cited as the responsible factors in recanalization of the aneurysms treated by endovascular approach[20].

Regrowth of completely thrombosed giant aneurysm has been reported and various mechanisms have been proposed including accumulation of thrombotic material, recurrent intramural hemorrhage or development of intrathrombotic capillary channels[21-24].

However, the phenomenon of recanalization of spontaneously thrombosed aneurysm is less well understood with few such reports[25-28]. Vasospasm[26,27] and systemic hypotension[28] were postulated as possible mechanisms for nonvisualisation of the aneurysms in these reports.

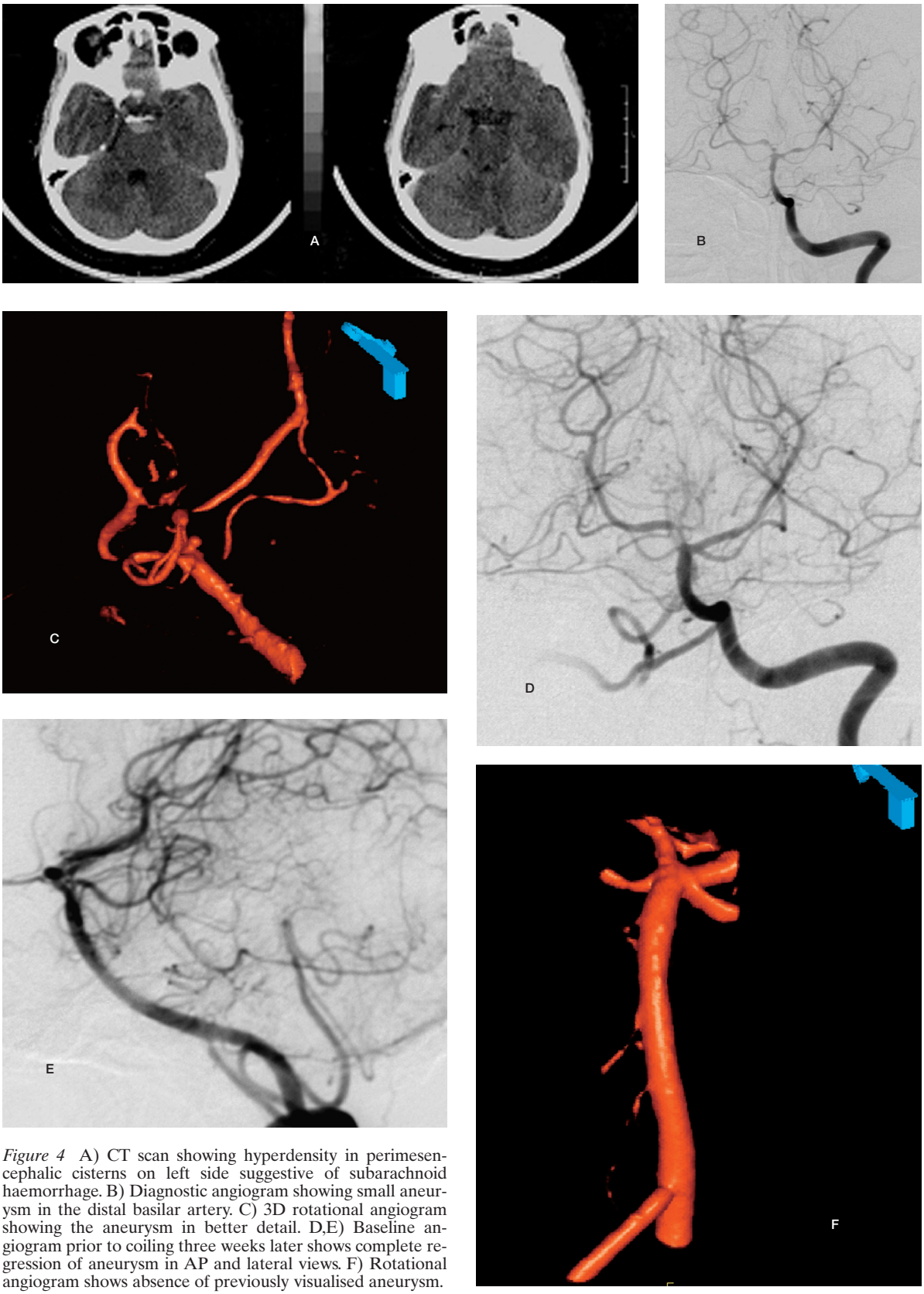

Figure 4 A) CT scan showing hyperdensity in perimesencephalic cisterns on left side suggestive of subarachnoid haemorrhage. B) Diagnostic angiogram showing small aneurysm in the distal basilar artery. C) 3D rotational angiogram showing the aneurysm in better detail. D,E) Baseline angiogram prior to coiling three weeks later shows complete regression of aneurysm in AP and lateral views. F) Rotational angiogram shows absence of previously visualised aneurysm.

Combined effects of clot organization and clot retraction may be causal factors for the reappearance of a spontaneously thrombosed aneurysm.

Nakajima et Al[25] suggested that the narrow, long neck of the aneurysm and dense clot in the neck of the aneurysm may have been responsible for temporary non visualisation of the aneurysm.

Conclusions

The decision to follow-up patients after noting a regression in size of the aneurysms was important. Hence, evolution of cerebral aneurysm should be considered a dynamic process and thrombosis should be considered a possibility in cerebral aneurysms which change in morphology.

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